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GLYCOPROTEIN DEPOSITION IN VASCULAR WALLS OF DIABETIC RETINOPATHY STUDIED BY IMMUNOHISTOCHEMISTRY

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Purpose: Proliferative diabetic retinopathy is assumed to develop because vasogenic factors are released from retinal areas that are ischaemic and hypoxic secondary to occlusion of the retinal vascular bed. It has been proposed that vascular occlusion in diabetic retinopathy may be due to the deposition of periodic acid Schiff positive glycoprotein compounds in the retinal vascular walls, but the role of these glycoproteins for retinal vascular occlusion has not been established.
Methods: Periodic acid Schiff staining and immunohistochemistry to laminin, fibronectin, vitronectin and type VI collagen was studied in eleven areas of capillary closure and eleven adjacent control areas from seven eyes of five diabetic patients, and in five midperipheral areas from five eyes of five age-matched controls. In each eye from diabetic patients the localized vascular lesions were identified from casts of the retinal vascular system.
Results: In the retina from diabetic patients there was a significantly higher number of vessels showing periodic acid Schiff staining and immunoreactivity to the studied glycoproteins. However, there was no difference between the number of glycoprotein containing vessels in areas of vascular occlusion and in adjacent control areas in diabetic patients, and the material occluding the lumen centrally in areas of vascular occlusion did not show any glycoprotein staining or immunoreactivity.
Conclusions: The findings confirm that glycoproteins are deposited in the vascular walls of diabetic retinopathy, but the findings also suggest that other factors are involved in causing vascular occlusion in this disease.

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RETINOPATHY IN IDDM IN EUROPE, THE EURODIAB IDDM COMPLICATIONS STUDY.

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Purpose To asses the frequency of retinopathy in IDDM, and investigate potentially modifiable risk factors.

Methods In a multicentre, cross-sectional study of 3250 IDDM patients from 31 European diabetes centres retinopathy was assessed and related to Hb-A_{1c}, albumin excretion rate, blood pressure, cholesterol, triglyceride and fibrinogen. Retinopathy was evaluated by centrally graded retinal photographs. Laboratory values were assessed centrally. Blood pressure was measured with random zero sphygmomanometer.

Results Non-proliferative retinopathy was present in 35.6%, proliferative in 10.6% of patients. Significant risk factors for moderate/severe non-proliferative retinopathy were blood pressure and triglyceride, and for proliferative retinopathy triglyceride and fibrinogen.

Conclusions Vision threatening retinopathy is a common complication in IDDM. Apart from glycemic control, several other potentially modifiable risk factors may be important, such as raised blood pressure, plasma triglyceride and fibrinogen.

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EOG CHANGES IN NON-PROLIFERATIVE AND PROLIFERATIVE DIABETIC RETINOPATHY.

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Purpose. The aim of our study was to clarify the EOG changes in proliferative (PDR) and non-proliferative (NPDR) diabetic retinopathy.
Methods. The EOG procedure used was close to that described by Arden et al. (1962)[1]. 85 diabetic patients (141 eyes) were tested (97 eyes of patients with NPDR and 44 eyes of patients with PDR). On all patients fluorescein angiography was performed prior the EOG testing. 16 healthy subjects (32 eyes) served as a control group. Arden index, implicit time of the dark trough (DT) and implicit time of the light peak (LP) were evaluated.. Modifications of the t-test and linear correlation test were used.
Results. When compared with the values of the control group, the values of the Arden index and LP in NPDR presented significant differences ($p < 0.002$). The same was present between control group and PDR eyes ($p < 0.001$). The DT values did not show any difference in both NPDR and PDR eyes compared with those of healthy eyes. When parameters were compared between NPDR and PDR eyes, only Arden index values showed significant differences ($p < 0.001$). When linear correlation test was performed only NPDR eyes presented significant correlation between LP values and the extend of ischemia and oedema of the posterior pole.
Conclusions. The Arden index is the most sensitive indicator of EOG parameters when testing patients with diabetic retinopathy. In NPDR eyes, the implicit time of LP reflects the extend of some pathologic changes in the retina.

1. Arden G., A.Barrada, J. Kelsey. New clinical test of retinal function based upon the standing potential of the eye. Br.J.Ophth. 1962;46/;449-467.

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ADENOSINE IS A NEUROPROTECTIVE AGENT IN RETINAL ISCHAEMIA.

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A large body of evidence exists to show that adenosine receptor agonists act as neuroprotective agents in brain ischaemia (1). However, only a single preliminary study has been carried out to support such a role in the retina (2). In the present communication we provide detailed convincing evidence that adenosine receptor agonists can be used to attenuate the manifestations of ischaemia.
Ischaemia was induced to the rat retina by raising the intraocular pressure (120 mmHg) above the systolic blood pressure for 45 min. Three days after reperfusion the b-wave of the electroretiogram in these animals was reduced by 47 ± 5% (n= 13) compared with preischaemic values. In addition a change in the nature of the calretinin immunostaining pattern in the retina was apparent. The characteristic three bands of calretinin immunoreactivity in the inner plexiform layer were reduced to a single band. In addition morphometric analysis of plastic embedded retinal sections showed a significant "thinning" of the inner plexiform layer.
To examine the effect of adenosine agonists on retinal ischaemia, 2µl adenosine deaminase (ADA; 5U in vitreous humour) or the ADA inhibitor EHNA (250µM in vitreous humour) was injected into the vitreous humour just before delivering an ischaemic insult. In other animals the adenosine agonist R-PIA was injected i.p. (0.2mg/kg) just before ischaemia and once daily for the following two days. The results showed that EHNA and R-PIA were neuroprotective in that the b-wave of the electroretinogram was not reduced, the calretinin immunoreactivity was unchanged and no evidence for "thinning" in the inner plexiform layer was apparent. In contrast there was suggestive evidence for ADA exacerbates ischaemia.
[1] Rudolphi K.A. et al., TIPS 13, (1992) 439-445. [2] Rosenbaum P.S et al., Invest. Ophthal. Vis. Sci. 33, (1992) 1125.